period of SV40 carcinogenesis in our experiments. The role of ITR serum factor in vivo is still unknown. The appearance of ITR factor in sera of hamsters neonatally infected with SV40 may possibly reflect the process of primary carcinogenesis and may be favourable for tumor development. The specificity and nature of ITR serum factor remains to be examined. Apparantly, detection of ITR humoral factor at an early stage of carcinogenesis may provide a useful tool not only for a better understanding of the immunological aspects in viral-induced tumorigenesis, but also it may be of diagnostic and prognostic value.

On the turnover of exogenous ferritin in the cephalopod optic gland. A microprobe study[1]

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Summary. X-ray energy emission spectra of iron show that horse spleen ferritin, after injection into the blood of the octopus, is taken up, accumulated and disposed of again by lysosomes of the optic gland.

Electron microscopic evidence has been produced for the uptake of ferritin by the stellate (or main or chief) cells of the cephalopod optic gland[3], a presumed endocrine organ[4]. In the present note, we demonstrate by the use of energy dispersive X-ray analysis, that exogenous ferritin is accumulated transiently in lysosomes of the stellate cells with respect to its concentration in the blood.

8 octopuses (*Octopus vulgaris*), anaesthetized with 2% ethanol in seawater, were injected into the aorta with 1 g/kg of horse spleen ferritin (Fluka, twice cristallized, cadmium-free) and killed at intervals of 15 min, 1 h, 4 h and 24 h. The optic glands, pin head-sized organs, situated upon the optic tracts, were fixed in 2% osmic acid (in a cacodylate-buffered Ringer of 1180 mosm) and processed for conventional electron microscopy. Unstained sections of approximately 1000 Å thickness, mounted upon plastic grids, were examined in a LINK EDX 290 microprobe analyser, attached to a Zeiss EM 10.

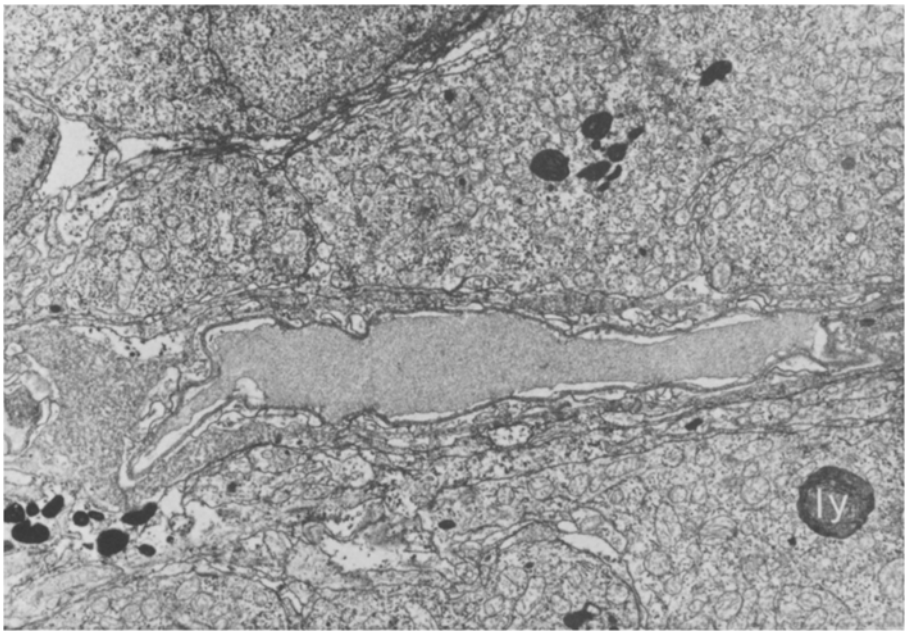

Fig. 1. Survey electron micrograph of an optic gland capillary of an octopus, injected with 1 g/kg of ferritin 1 h before fixation. This protein is taken up and disposed of again by the sort of lysosomes (ly) which are shown in the adjacent stellate cells. ×5460.

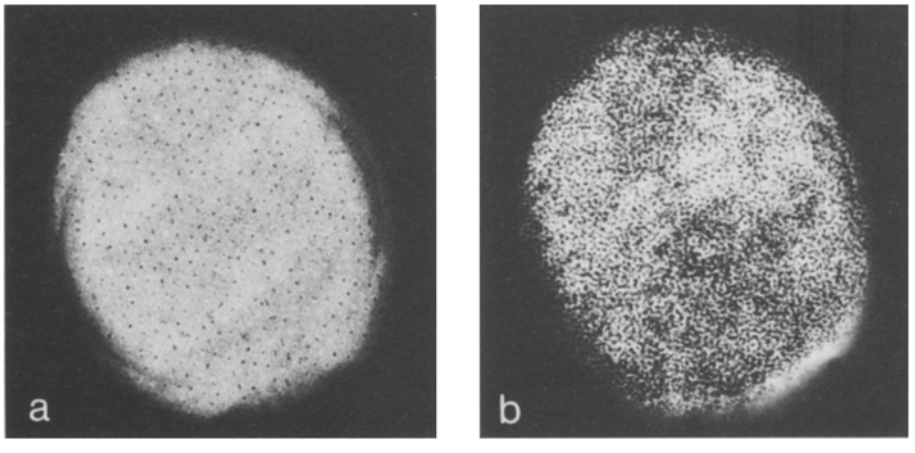

Fig. 2. Electron micrographs of (*a*) a blood vessel and (*b*) an optic gland lysosome, taken during the X-ray analysis. The blood vessel was fixed 1 h and the lysosome 4 h after the injection of ferritin. The ferritin molecules form a mixed crystal with the hemocyanin molecules in the blood of the octopus. In both micrographs, the diameter of the beam is 0.7 μm.

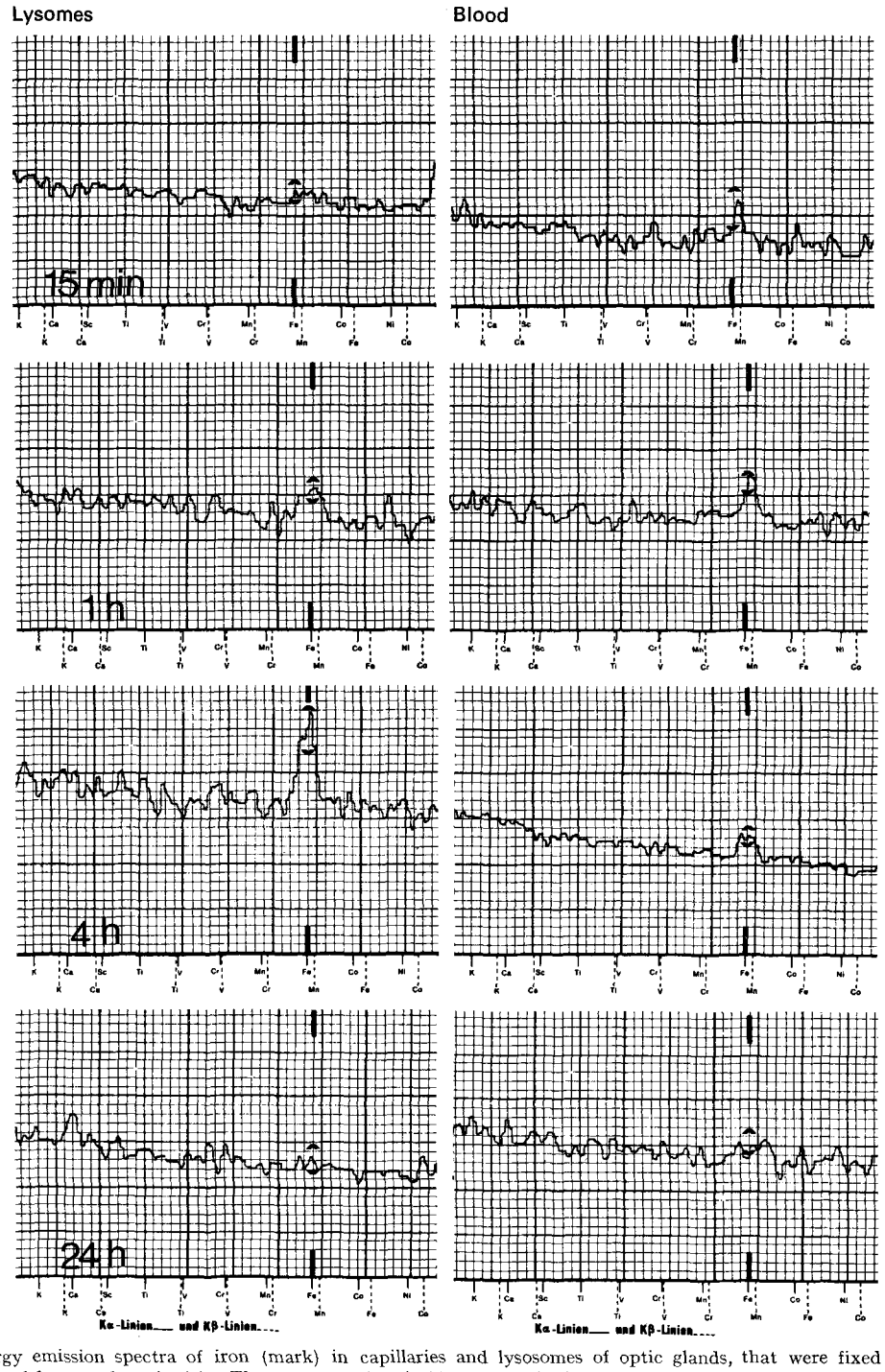

Fig. 3. X-ray energy emission spectra of iron (mark) in capillaries and lysosomes of optic glands, that were fixed at intervals after the administration of horse spleen ferritin. The exposure time is 90 sec and the beam current 40 μA. Maxima and minima of 5–8 different lysosomes (and blood vessels respectively) are indicated by arrowheads.

Horse spleen ferritin has a mol. wt of 800,000 and a particle size of 55 Å in electron micrographs. When circulating in the blood capillaries, the iron, which accounts for at least 20% of the mass of the ferritin molecule (according to the manufacturer), exhibited a low energy emission up to 4 h after the injection (figures 2 and 3), One day after the administration, the concentration of ferritin in the blood had fallen below the sensitivity of the method.

Ferritin molecules leave the capillaries rapidly and enter the chief cells by a mechanism as yet unknown[3]. They are taken up by lysosomes, which are extraordinarily abundant in these cells[5].

The pattern of ferritin activity in chief cell lysosomes was different from that in the blood: no emission at 15 min, increasing emission between 1 and 4 h, low emission again at the end of 24 h (figure 3). We do not know the mechanism by which ferritin is disposed of by the lysosomes. The evidence for a turnover of ferritin in the optic gland cells supports our hypothesis that the organ is involved in a catabolic function[3,5].

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